Dietary Fat and Breast Cancer in the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study [1]

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ABSTRACT—The relationship between dietary fat intake and breast cancer incidence was examined in the National Health and Nutrition Examination Survey I (NHANES I) Epidemiologic Follow-up Study cohort. This cohort is derived from adults (≥25 yr) examined in the NHANES I (1970–75) cross-sectional survey of the U.S. population and provides a mean follow-up time of 10 years. An analytic sample of 5,485 women, including 99 breast cancer cases (34 premenopausal and 65 postmenopausal at NHANES I baseline), was examined for associations with dietary intake of fat, percent energy from fat, total energy, saturated fat, polyunsaturated fat, monounsaturated fat, and cholesterol on the basis of a 24-hour recall administered at the baseline NHANES I examination. No significant differences in dietary fat intake between cases and noncases were evident when mean intakes for each group were compared. For total fat (g) and saturated fat (g), a significant inverse association was indicated in proportional hazards analyses. Adjustment of fat for total energy intake resulted in a smaller effect that was no longer statistically significant. Adjustment for accepted breast cancer risk factors did not change these findings. This prospective study of a sample from the U.S. population does not support the hypothesis that high dietary fat intake increases breast cancer risk. Indeed, some lower risk associated with high fat intake may be indicated, although this result may be influenced by methodologic problems with the dietary assessment.—JNCI 1987; 79:465–471.

Both laboratory (*1, 2*) and epidemiologic (*3–13*) evidence suggest that a high intake of fat can increase susceptibility to breast cancer. However, the evidence is far from conclusive. Recent experimental studies (*14, 15*) emphasize the strong correlation between fat and total energy intake and indicate that, when both factors are considered together, total energy intake appears to be the stronger indicator of risk. In human studies the strongest evidence for fat as a risk factor for breast cancer comes from international correlation studies (*3–8*) and within-country descriptive studies (*9–11*). Results of case-control studies have been equivocal. Of four such studies in which fat intake could be quantified (*12, 16–18*), only one (*12*) found any significant association with dietary fat, and in that study intake of all nutrients reported was higher in the cases. Several other studies have examined breast cancer in relation to consumption of certain foods or food groups high in fat, with similarly equivocal results. Positive associations with meat (*19, 20*) have been reported, yet vegetarian nuns did not differ from other single women in breast cancer mortality (*21*). Associations with fried potatoes (*22*), dairy products (*23*), cheese (*24*), and generally high-fat foods (*25*) have been noted, but none with butter (*24*) or fats and oils (*26*). To our knowledge, three prospective studies have been carried out (*13, 27, 28*). The first (*13*) involved 10 years of follow-up of 142,857 Japanese females and 142 breast cancer deaths. Although the dietary assessment did not allow estimation of total fat intake, women reporting daily intake of meat showed an elevated standardized mortality ratio compared to that seen in occasional meat eaters and non-meat eaters of 1.26 in women 40–54 years of age and 2.38 in women age 55 or older. In a cohort of Seventh-Day Adventist women followed for 21 years, however, no significant association between meat consumption and breast cancer mortality was evident (*27*). Most recently, Willett et al. (*28*) have shown no positive association between dietary fat and breast cancer incidence in a cohort of 89,538 women with 601 cases of breast cancer. Recent data from the NCHS provide another opportunity to examine prospectively the association between dietary fat intake and breast cancer incidence in a large cohort of American women.

SUBJECTS AND METHODS

NHEFS design.—NHANES I and its Augmentation Survey were conducted by NCHS from 1971 to 1975 (*29, 30*). These surveys provided cross-sectional information on demographic, nutritional, biochemical, clinical, anthropometric, and medical history variables in a sample selected to represent the U.S. noninstitutionalized

ABBREVIATIONS USED: BMI=body mass index; CI=confidence interval; kcal=kilocalories; NCHS=National Center for Health Statistics; NHANES I=National Health and Nutrition Examination Survey I; NHEFS=NHANES I Epidemiologic Follow-up Study; PIR=poverty index ratio; RR=relative risk.

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population. Certain population groups were oversampled: children 1-5 years, women 20-44 years, the elderly ≥65 years, and low-income individuals. In January 1979, planning began for a follow-up study of the 14,407 adults (≥25 yr old) examined in NHANES I. Details of the study design are provided elsewhere (*31*). Data from the initial follow-up period (May 7, 1982, through August 15, 1984) were provided by NCHS for analysis. The mean interval of follow-up was 10 years. The follow-up data include interview information from subjects or proxies, weight and blood pressure measurements, hospital and nursing home records, and death certificate information.

Study population.—The NHEFS cohort consists of 14,407 people aged 25-74 years at the time of their examination for NHANES I, of whom 8,596 (59.7%) were women. As this analysis focused on dietary variables, women with inadequate or unreliable baseline dietary data were excluded. These exclusions included women with no dietary data ($n=1,727$), women whose dietary data were obtained from a proxy ($n=205$), women whose dietary information was considered "unsatisfactory" by the nutritionist collecting the data ($n=35$), and women with imputed data ($n=117$). Women pregnant at the time of or within the 3 months prior to baseline ($n=238$) and women who were breast-feeding ($n=6$) were excluded because their dietary data were not considered appropriate for comparison. Seven women were excluded because of indications of prevalent breast cancer at baseline. An additional group of 776 women was lost to follow-up either through the inability to trace them or their refusal to participate. After all exclusions, the final analytic cohort consisted of 5,485 women.

Determination of cases.—Cancer cases were identified at the time of follow-up from self-reports by individuals or proxies, hospital records, and/or death certificates. Because of potential confusion between benign and malignant breast diseases, only breast cancer cases identified through either hospital records or death certificates were considered confirmed and used in this analysis. Self-reported cases without confirmation were grouped with non-cases for analyses. A total of 99 confirmed cases were identified, 84 of whom had hospital diagnoses and 15 with only death certificate information. We computed the expected number of breast cancer cases in this cohort based on age- and race-specific incidence rates from the Connecticut Cancer Registry and compared the expected to the observed number of cases. The ratio of observed-to-expected breast cancer cases was 0.93, with a 95% CI of 0.75-1.13, providing reassurance that case ascertainment was reasonably complete. Menopausal status of the cases at the time of the baseline NHANES I examination was 34 premenopausal and 65 postmenopausal. Within the group of confirmed breast cancer cases, the date of the first hospitalization with breast cancer was taken as the onset date. When only death certificate confirmation was available, the date of death was considered the date of onset.

Dietary variables.—Dietary data used in this analysis were obtained at the baseline NHANES I examination and were based on a 24-hour recall conducted by a trained nutritionist using three-dimensional graduated food portion models. The 24-hour recall data were analyzed for nutrient composition using a program developed at Tulane University based on food composition data from the U.S. Department of Agriculture, industry, and other sources (*32*). The dietary variables from the 24-hour recall analyzed in this study were fat, percent energy from fat, total energy intake, saturated fat, polyunsaturated fat, monounsaturated fat, and cholesterol intake. Ten persons had missing data for fatty acid and cholesterol intake and are therefore missing from all analyses of these dietary variables. Details on the procedures for the dietary examination have been documented (*33*).

Statistical analysis.—The effect of dietary fat on the incidence of breast cancer was assessed in two ways: 1) comparing adjusted means of dietary intakes from women who developed breast cancer during the follow-up period with those who did not and 2) using proportional hazards survival analysis methods available on the SAS statistical software package (*34*). To compute mean fat intakes adjusted for age and other breast cancer risk factors, the dietary variables were treated as dependent variables in general linear regressions with the adjustment factors as independent variables and an indicator variable to identify case status. Since all the dietary variables except percent energy from fat were right skewed, analyses were repeated using log transformed values. As similar results were obtained with the transformed values, only results from the untransformed data are shown here. For the proportional hazards analysis, dietary intake variables were split into quartiles based on their distribution in the analytic cohort ($n=5,485$). The analytic strategy was to develop a basic model for breast cancer, incorporating currently suggested risk factors in the most efficient manner, and then to add the dietary variables of interest to this model. Education, PIR (a measure of adjusted income computed by NCHS), BMI (kg/m^2), parity, age at menarche, menopausal status-age at menopause, age at first birth, and family history of breast cancer were each examined separately in bivariate regressions with age at the baseline examination to determine their contribution to breast cancer risk in this sample. Age was modeled as a trend variable having values 1, 2, 3, and 4, corresponding to the four categories 25-34, 35-54, 55-64, and ≥65 years. Analyses were repeated using age as a continuous variable, and results were similar. Indicator variables were used to model the other risk factors to avoid the necessary linearity assumptions of continuous or trend variables. For PIR and BMI, the categories chosen were data derived and represent the upper quintile where breast cancer risk appeared to concentrate (PIR ≥3.75 vs. PIR <3.75; BMI ≥30.0 vs. BMI <30.0). For the other variables, categories frequently presented in breast cancer analyses were chosen: education, >high school versus ≤high school; parity, 1-2, ≥3 children versus nulliparous; age at menarche, 12-13, ≥14 years versus <12 years; menopausal status-age at menopause, premenopausal, postmenopausal >45 years

versus postmenopausal ≤45 years; age at first birth, 20-24, 25-29, ≥30 years versus <20 years; and family history of breast cancer, yes versus no in a first-degree relative. Based on a step-down analysis from the full model, only five of the eight variables (PIR, BMI, age at menarche, premenopausal status-age at menopause, and family history) were significantly associated with breast cancer and were kept in a model with age to examine the dietary variables. The age-adjusted distribution of these five factors across quartiles of dietary fat intake was examined. Age adjustment was carried out using direct adjustment to 10-year age cuts of the analytic cohort. Information on family history and age at first birth was available only at the follow-up interview. All other variables were assessed at baseline. Benign breast disease could not be examined because no adequate history of it was elicited either at baseline or at follow-up. Stratified analyses were run to examine the fat association by menopausal status.

RESULTS

The standard breast cancer risk factors generally showed the expected associations. The socioeconomic indicator variable, PIR ≥3.75, was significantly positively related to breast cancer incidence (RR=1.8). The upper quartile of fatness (BMI ≥=30) also showed a significant elevation in risk (RR=1.7). An older age at menopause (>45 yr) increased risk (RR=1.4, not significant), as did a family history of breast cancer in a first-degree relative (RR=2.0). An older age at menarche increased risk compared to menarche at 11 years or younger (RR=3.3 for menarche at 12, 13 yr; RR=2.8 for menarche at ≥14 yr), a finding not usually observed. However, including age 12 in the reference group eliminated any significant effect of age at menarche. Because of this anomalous finding, the multivariate analyses were rerun excluding this variable, but results were similar.

The distribution of age groups and the age-adjusted distribution of the significant breast cancer risk factors across dietary fat quartiles are shown in table 1. There was a strong trend for younger women to report total fat intakes in the upper quartiles and older women to concentrate in the lower quartiles. This pattern reflected total energy intake differences across the age groups as well, as expressing fat intake as a percentage of energy weakened the trend considerably. Women with high relative income, PIR ≥3.75, and premenopausal women were disproportionately in the higher fat intake quartiles. Fatter women, BMI ≥30.0, had total fat intakes concentrated in the two lower quartiles. Neither age at menarche nor family history exhibited any association with fat intake. When fat was expressed relative to energy intake (% kcal), no associations were evident for these risk factors.

A summary of the adjusted mean daily nutrient intakes for the breast cancer cases and controls is given in table 2. The mean intake for cases was less than that for non-cases for all the dietary variables examined, but the differences were not statistically significant. Further adjustment for standard risk factors resulted in slightly greater differences between cases and non-cases, although not enough to achieve statistical significance.

The adjusted RRs associated with quartiles of dietary fat and energy intake are presented in table 3. The upper quartile of fat and saturated fat showed a significant protective effect compared to the effect seen with the lowest quartile of these variables. A significant trend in risk over the quartiles was seen for fat and saturated fat in the full proportional hazards model. Fat as a percentage of energy had a marginally significant trend across the quartiles. None of the other dietary fat and energy variables showed any significant relationship with breast cancer incidence. Adjustment for the significant standard breast cancer risk factors in this sample did not substantially alter any of the observed associations, although (as in the comparison of mean intakes) it slightly strengthened the difference in most cases.

Stratified analyses showed that the apparent protective effect of high fat intake was strongest in premenopausal women (RR=0.08; 95% CI=0.01-0.61) and no longer statistically significant in postmenopausal women, despite the larger number of cases in the postmenopausal group (RR=0.63; 95% CI=0.30-1.54).

TABLE 1.—*Age-adjusted distribution of breast cancer risk factors across quartiles of dietary fat intake*

Variable	Fat, g/day, quartiles[a]				Fat, % kcal/day, quartiles[a]			
	<38	38-53.9	54-73.9	≥=74	<30	30-35.9	36-41.9	≥=42
Age, yr								
25-34	19	23	25	33	23	26	27	24
35-54	21	22	26	31	21	25	28	26
55-64	30	26	24	20	26	28	23	23
≥65	33	28	24	15	28	27	24	21
PIR, ≥3.75, top quartile	20	26	25	29	23	29	26	22
BMI, ≥30.0, top quartile	32	26	21	21	26	25	24	25
Age at menarche, <12 yr	24	25	27	24	24	27	25	24
Premenopausal	18	23	29	30	24	22	32	22
Family history of breast cancer[b]	22	26	27	25	26	27	25	22

[a] Results in cols. are percents.
[b] First-degree relative.

DISCUSSION

In this prospective study no significant difference in mean intake of various measures of dietary fat was observed between women who developed breast cancer and those who did not. However, when individuals were examined with the use of proportional hazards techniques, a decrease in risk for the highest quartile of dietary fat and saturated fat intake was observed. Although a prospective study such as the NHEFS avoids the problems of recall bias found in case-control studies, it is still hampered by the fact that the 24-hour recall does not adequately depict the individual's usual pattern of intake. The problem of appropriate dietary instruments for use in epidemiologic studies has been reviewed in detail (*35*). Most nutritionists agree that the 24-hour recall method used in NHANES I provides an unbiased estimate of a group's mean nutrient intake. The proportional hazards techniques, which are best suited statistically for analyzing risk factors in prospective data, must be interpreted with caution due to the use of 24-hour recall data as if they adequately depicted an individual's usual intake. The resulting misclassification tends to bias results toward the null.

Recently, Willett et al. (*28*) reported no positive association between dietary fat and breast cancer in their analysis of 4 years' follow-up in a prospective cohort of 89,538 women. In their study the highest risk occurred in the group representing the lowest quintile of fat intake. Other researchers have found an inverse association with dietary fat intake and colorectal cancer in a prospective study (*36*). Colorectal cancer incidence and breast cancer incidence are typically highly correlated in ecologic data; therefore, these colorectal findings are pertinent. Stemmermann et al. (*36*) concluded their report by suggesting that "the balance between energy consumption and expenditure might be more important than the amount or types of nutrients consumed." Confounding effects of energy balance may also be relevant here.

The validity of the NHANES I 24-hour recall data has been questioned because of the low energy intakes reported in the face of an increasingly overweight population (*37*). The 1,400-kcal mean energy intake seen in this sample is comparable to values seen in other surveys of the U.S. population, such as the 1977-78 Nationwide Food Consumption Survey (*38*) and the National Health and Nutrition Examination Survey II, 1976-80 (*39*). Such low energy intakes, whether real or underreported, are important for this analysis only if there is differential underreporting according to breast cancer status. Braitman et al. (*40*) also reported a slight negative association between obesity and energy intake in women in the NHANES I sample and suggested that differential underreporting of intake by the obese could be one cause of their findings. However, they considered it an unlikely explanation because of the probing, use of models, and standardization techniques used for dietary assessment in NHANES I. Nevertheless, if such underreporting occurred or if breast cancer was diagnosed earlier in leaner women, it would work to obscure any positive association between fat intake and breast cancer, since breast cancer risk is increased in obese women in these data. Underreporting by the obese in conjunction with true higher fat intakes in leaner and presumably more active women could produce a negative association such as indicated here in the proportional hazards analyses. To examine this possible explanation, the analyses were repeated within tertiles of BMI. If underreporting by the fatter women was the explanation, one would expect no dietary fat association to be evident in the leaner tertiles. However, a similar pattern of lower RR associated with high fat intake was seen within each BMI tertile (BMI $\leq$22.0 with RR=0.65 and 95% CI=0.17-2.52; 22.1$\leq$ BMI $\leq$28.0 with RR=0.08 and 95% CI=0.01-0.65; BMI $\geq$28.1 with RR=0.41 and 95% CI=0.11-1.47).

Expressing fat as a percentage of calories is one way of correcting for total body size and energy requirement. Other means of adjusting total fat intake for body size and energy intake include examining fat per kilogram body weight and examining residuals of fat after regressing kcal. Both approaches yielded similar results to fat as a percentage of calories (RRs for upper quartile compared to lowest quartile, adjusted for age and other breast cancer risk factors, of 0.66 for fat/kg body wt and 0.68 for fat residuals, not statistically significant). The absence of a significant negative association for fat when adjusted for indicators of energy requirement (caloric

TABLE 2.—*Mean (SEM) daily nutrient intakes for breast cancer cases and non-cases adjusted for age and for breast cancer risk factors in this sample*[a]

Nutrient (units)	Age-adjusted		Full model[b]	
	Cases (n=99)	Non-cases (n=5,386)	Cases (n=86)	Non-cases (n=4,912)
Fat (g)	57.0 (3.2)	59.9 (0.4)	55.0 (3.4)	60.3 (0.5)
Fat (% energy)	34.6 (0.9)	36.0 (0.1)	34.6 (0.9)	36.0 (0.1)
Energy (kcal)	1,441 (61)	1,465 (8)	1,404 (65)	1,475 (9)
Saturated fat (g)	20.0 (1.3)	21.4 (0.2)	19.4 (1.4)	21.5 (0.2)
Polyunsaturated fat (g)	6.6 (0.6)	6.6 (0.1)	6.1 (0.6)	6.7 (0.1)
Monounsaturated fat (g)	21.8 (1.3)	22.9 (0.2)	21.0 (1.4)	23.1 (0.2)
Cholesterol (mg)	282 (24)	305 (3)	268 (26)	305 (3)

[a] No statistically significant differences were found in any of these nutrient comparisons.
[b] Model including age, PIR, BMI, age at menarche, menopausal status-age at menopause, and family history of breast cancer. Analyses done on subset of women with complete information.

TABLE 3.—*RRs for breast cancer incidence by quartiles of dietary fat and energy intake adjusted for age and for breast cancer risk factors in this sample*

Variable	Age-adjusted			Full model[a]		
	Cases (n=99)	Non-cases (n=5,386)	RR (95% CI)[b]	Cases (n=86)	Non-cases (n=4,912)	RR (95% CI)[b]
Fat, g						
<38	33	1,337	1.00	29	1,198	1.00
38-53.9	24	1,313	0.78 (0.46-1.33)	21	1,194	0.73 (0.42-1.29)
54-73.9	29	1,350	0.95 (0.58-1.58)	27	1,234	0.96 (0.57-1.63)
≥74	13	1,386	0.47 (0.25-0.91)	9	1,286	0.34 (0.16-0.73)
			P for trend=.07			*P* for trend=.03
Fat, % energy						
<30	26	1,277	1.00	22	1,157	1.00
30-35.9	38	1,397	1.38 (0.84-2.27)	35	1,279	1.50 (0.88-2.56)
36-41.9	20	1,403	0.77 (0.43-1.38)	16	1,292	0.73 (0.38-1.38)
≥42	15	1,309	0.62 (0.33-1.19)	13	1,184	0.66 (0.33-1.31)
			P for trend=.05			*P* for trend=.06
Energy, kcal						
<1,030	26	1,338	1.00	23	1,193	1.00
1,030-1,378.9	31	1,337	1.23 (0.73-2.08)	28	1,209	1.23 (0.71-2.13)
1,379-1,775.9	24	1,349	0.99 (0.57-1.71)	21	1,260	0.89 (0.49-1.63)
≥1,776	18	1,362	0.87 (0.47-1.61)	14	1,250	0.70 (0.36-1.40)
			P for trend=.54			*P* for trend=.22
Saturated fat, g						
<13	34	1,431	1.00	29	1,282	1.00
13-18.9	23	1,275	0.81 (0.47-1.37)	21	1,176	0.83 (0.47-1.45)
19-26.9	30	1,287	1.07 (0.65-1.76)	29	1,172	1.18 (0.70-1.98)
≥27	12	1,383	0.44 (0.23-0.86)	7	1,272	0.29 (0.12-0.67)
			P for trend=.07			*P* for trend=.04
Polyunsaturated fat, g						
<3	31	1,398	1.00	27	1,229	1.00
3-4.9	19	1,144	0.78 (0.44-1.37)	17	1,065	0.75 (0.41-1.38)
5-8.9	28	1,555	0.90 (0.54-1.50)	26	1,423	0.93 (0.54-1.59)
≥9	21	1,279	0.93 (0.53-1.63)	16	1,185	0.73 (0.39-1.36)
			P for trend=.85			*P* for trend=.45
Monounsaturated fat, g						
<14	31	1,365	1.00	28	1,225	1.00
14-19.9	24	1,222	0.90 (0.53-1.53)	20	1,124	0.82 (0.46-1.45)
20-28.9	25	1,451	0.81 (0.48-1.38)	24	1,318	0.83 (0.48-1.43)
≥29	19	1,338	0.74 (0.41-1.34)	14	1,235	0.59 (0.30-1.13)
			P for trend=.28			*P* for trend=.14
Cholesterol, mg						
<130	25	1,333	1.00	22	1,197	1.00
130-232.9	31	1,321	1.29 (0.76-2.18)	30	1,221	1.33 (0.76-2.31)
233-414.9	24	1,370	0.95 (0.54-1.66)	19	1,261	0.79 (0.43-1.46)
≥415	19	1,352	0.80 (0.44-1.47)	15	1,223	0.70 (0.36-1.37)
			P for trend=.32			*P* for trend=.12

[a] Model including age, PIR, BMI, age at menarche, menopausal status-age at menopause, and family history of breast cancer. Analyses done on subset of women with complete information.
[b] RRs (95% CIs) for proportional hazards model.

intake, body wt) suggests that total energy balance is also relevant, although kcal alone were not significantly associated with breast cancer risk. However, with only 99 cases, this study has low power to detect significant differences.

Another possible explanation of the result found here is the substitution of alcohol for fat calories in the diet. Alcohol intake has been associated with an increased risk for breast cancer in other studies (*41*) and is a significant risk factor in this sample (*42*). However, total fat intake (g) is positively associated with alcohol intake in this sample, and fat as a percentage of calories is only weakly inversely associated with alcohol. Thus controlling for alcohol intake did not reduce the fat association.

It should be noted that the distribution of dietary fat intakes examined in this analysis is heavily shifted toward relatively high intakes. Thus the disparity between these findings and cross-cultural studies may be because fat exerts an effect only up to a certain threshold level (e.g., 15 or 25% of energy), which could not be addressed in this sample. It may also be that dietary fat intake exerts its major influence early in life, such as during puberty, and an analysis of diets in adult women is not relevant. In addition, the influence of any dietary changes that may have occurred after the baseline dietary assessment could not be determined in these analyses.

These results could have been affected by differing

influences of dietary fat on incidence and on survival, since for 15 breast cancer cases date of death was used as date of incidence. To check this possibility, analyses were rerun, separating out the 15 cases determined from death certificates. Although numbers were smaller, the same inverse association with high fat intake was seen in cases determined by death certificates as in those ascertained from hospital diagnoses. The use of only hospital or death certificate-confirmed cases in this analysis introduces a potential for bias if such case confirmation was linked in any way with dietary intake. Such a link is unlikely, and repeating the analyses including self-reports as cases resulted in findings similar to those reported here. These results could also be biased if the women lost to follow-up (some of whom would probably be cases) substantially differed in their dietary intake from the analytic cohort. However, a comparison between the baseline dietary intake and other descriptive characteristics of the lost-to-follow-up group and the analytic cohort revealed no significant differences.

This study is an important addition to the examination of diet and breast cancer because of its prospective nature in which careful dietary assessment was carried out prior to ascertainment of breast cancer status. The NHANES I survey that provided this cohort sampled from a large cross-section of the U.S. population, and thus these data do not focus primarily on the upper-socioeconomic-class women at higher risk of breast cancer who are usually the focus of breast cancer studies. These data are not consistent with the hypothesis that high dietary fat intake increases breast cancer risk. Indeed, they suggest a possible protective effect of high fat intake, but this result may be influenced by methodologic problems with the dietary assessment. These results certainly indicate the need for further exploration.

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